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 NEWS  1          Web Page for STN Seminar Schedule - N. America
 NEWS  2  AUG 10  Time limit for inactive STN sessions doubles to 40
                  minutes
 NEWS  3  AUG 18  COMPENDEX indexing changed for the Corporate Source
                  (CS) field
 NEWS  4  AUG 24  ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
 NEWS  5  AUG 24  CA/CAplus enhanced with legal status information for
                  U.S. patents
 NEWS  6  SEP 09  50 Millionth Unique Chemical Substance Recorded in
                  CAS REGISTRY
 NEWS  7  SEP 11  WPIDS, WPINDEX, and WPIX now include Japanese FTERM
                  thesaurus
 NEWS  8  OCT 21  Derwent World Patents Index Coverage of Indian and
                  Taiwanese Content Expanded
 NEWS  9  OCT 21  Derwent World Patents Index enhanced with human
                  translated claims for Chinese Applications and
                  Utility Models
 NEWS 10  OCT 27  Free display of legal status information in CA/CAplus,
                  USPATFULL, and USPAT2 in the month of November.

 NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,
              AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

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FILE 'HOME' ENTERED AT 11:21:01 ON 09 NOV 2009

=> file medline biosis caplus embase
COST IN U.S. DOLLARS                                 SINCE FILE      TOTAL
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FULL ESTIMATED COST                                      0.44       0.44

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=> s insulin(w)receptor(w)substrate(w)2 or IRS(w)2 and (activator or inhibitor)
L1        3541 INSULIN(W) RECEPTOR(W) SUBSTRATE(W) 2 OR IRS(W) 2 AND (ACTIVATOR
               OR INHIBITOR)

=> s l1 and (over(w)express or over(w)produce)
L2           2 L1 AND (OVER(W) EXPRESS OR OVER(W) PRODUCE)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3           2 DUP REM L2 (0 DUPLICATES REMOVED)

=> dis ibib abs l3

L3   ANSWER 1 OF 2  EMBASE  COPYRIGHT (c) 2009 Elsevier B.V. All rights
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ACCESSION NUMBER:   2005199769  EMBASE
TITLE:              The GLUTs family - Lessons from transgenic mice.
AUTHOR:             Hartil, K.; Weldon, R.H.; Seki, Y.; Charron, M.J.
                    (correspondence)
CORPORATE SOURCE:   Department of Biochemistry, Albert Einstein College of
                    Medicine, 1300 Morris Park Avenue, Bronx, NY 10461, United
                    States. charron@aecom.yu.edu
SOURCE:             Current Medicinal Chemistry: Immunology, Endocrine and
                    Metabolic Agents, (Apr 2005) Vol. 5, No. 2, pp. 189-206.
                    Refs: 144
                    ISSN: 1568-0134  CODEN: CMCIC8
COUNTRY:            Netherlands
DOCUMENT TYPE:      Journal; General Review; (Review)
FILE SEGMENT:       016     Cancer
                    018     Cardiovascular Diseases and Cardiovascular Surgery
                    022     Human Genetics
                    029     Clinical and Experimental Biochemistry
                    003     Endocrinology
                    005     General Pathology and Pathological Anatomy
LANGUAGE:           English
SUMMARY LANGUAGE:   English
ENTRY DATE:         Entered STN: 19 May 2005
                    Last Updated on STN: 19 May 2005
AB   The glucose transporters (GLUTs) are currently a 13 member family of
     facilitative transmembrane proteins which transport glucose down its
     concentration gradient.  The GLUTs have a tissue specific expression and
     regulation.  Dysregulation of GLUTs have been implicated in the
     pathogenesis of a number of diseases including diabetes and cancer and are
     known to play an important role in the developing embryo.  In addition,
     roles for GLUTs in cardiac function and embryonic development have been
     identified and will be discussed in this review.  The ability to ablate or
     over-express GLUTs has advanced our understanding of the
     role these transporters play in the maintenance of normal glucose
     homeostasis and the pathogenesis of diabetes.  The development of Cre-LoxP
     technology coupled with the existence of tissue specific promoters allows
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investigators to manipulate gene expression both globally and in a tissue specific manner. The major GLUTs which have been investigated using transgenic technology are GLUT1, GLUT4 and GLUT2. Overexpression of GLUT4 and GLUT1 results in increased glucose uptake and metabolism. However, only GLUT4 overexpression protects against the development of insulin resistance in transgenic mice. Genetic ablation of GLUT4 and GLUT2 results in impaired insulin tolerance and defects in both lipid and glucose metabolism. This review will present various transgenic models of GLUT modification and discuss what has been learned from these models about the role that GLUTs play in glucose homeostasis, insulin action and development. .COPYRGT. 2005 Bentham Science Publishers Ltd.

=> dis ibib abs l3 2

L3 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:129407 CAPLUS
DOCUMENT NUMBER: 134:261420
TITLE: Specific inhibition by hGRB10ζ of insulin-induced glycogen synthase activation: evidence for a novel signaling pathway
AUTHOR(S): Mounier, C.; Lavoie, L.; Dumas, V.; Mohammad-Ali, K.; Wu, J.; Nantel, A.; Bergeron, J. J. M.; Thomas, D. Y.; Posner, B. I.
CORPORATE SOURCE: The Polypeptide Hormone Laboratory, McGill University, Montreal, QC, H3A 2B2, Can.
SOURCE: Molecular and Cellular Endocrinology (2001), 173(1-2), 15-27
CODEN: MCEND6; ISSN: 0303-7207
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Grb10 is a member of a family of adapter proteins that binds to tyrosine-phosphorylated receptors including the insulin receptor kinase (IRK). In this study recombinant adenovirus was used to over-express hGrb10ζ, a new Grb10 isoform, in primary rat hepatocytes and the consequences for insulin signaling were evaluated. Over-expression of hGrb10ζ resulted in 50% inhibition of insulin-stimulated IRK autophosphorylation and activation. Anal. of downstream events showed that hGrb10ζ over-expression specifically inhibits insulin-stimulated glycogen synthase (GS) activity and glycogen synthesis without affecting insulin-induced IRS1/2 phosphorylation, PI3-kinase activation, insulin like growth factor binding protein-1 (IGFBP-1) mRNA expression, and ERK1/2 MAP kinase activity. The classical pathway from PI3-kinase through Akt-PKB/GSK-3 leading to GS activation by insulin was also not affected by hGrb10ζ over-expression. These results indicate that hGrb10ζ inhibits a novel and presently unidentified insulin signaling pathway leading to GS activation in liver.
OS.CITING REF COUNT: 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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LOGOFF? (Y)/N/HOLD:y

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FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 11:22:01 ON 09 NOV 2009
L1 3541 SEA FILE=MFE SPE=ON ABB=ON PLU=ON INSULIN(W) RECEPTOR(W)

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                SUBSTRATE(W) 2 OR IRS(W) 2 AND (ACTIVATOR OR INHIBITOR)
L2            2 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  L1 AND (OVER(W) EXPRESS
                OR OVER(W) PRODUCE)
L3            2 DUP REM L2 (0 DUPLICATES REMOVED)
                DIS IBIB ABS L3
                DIS IBIB ABS L3 2
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    40.11      40.55

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                    -0.82      -0.82

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